We present here a discussion of the two main messages that emerged from the meeting.

MANY DISEASES IN HUMANS, INCLUDING RESPIRATORY DISEASES, HAVE COMMON MOLECULAR, GENETIC AND EPGENETIC ROOTS

Dr Batemen discussed the ACOS syndrome (asthma COPD overlap syndrome), which has recently been the subject of much study and controversy. Although ACOS shares clinical phenotype features with AB and COPD, it is not clear if it is a distinct disease entity or if it simply reflects the chance coexistence of 2 common diseases in the same patient. Genetic studies have so far failed to reach a consensus: some researchers have identified specific ACOS alterations, while others find these impossible to reproduce. Several oral communications (N. Larocca, A. Crespo, S. Pérez, R. Faner, C. Casadevall) addressed the issue of AB and/or COPD.

With regard to COPD-LC, Dr Spira presented his theory on the common “airway battlefield”. He suggested that tobacco has an impact, only partially reversible, on genetic (mRNA) and epigenetic (microRNA) expression throughout the respiratory epithelium, from the nose to the alveoli. This could be clinically relevant because this “molecular signature” could be detected even in an apparently normal epithelium using macro and microscopic techniques. This could be of use in the identification of smokers with a high risk of developing COPD and/or LC. Moreover, these molecular changes should be reversible, at least in part, with the use of inhaled corticosteroids. Dr P. Rivera talked at greater depth about aspects related with LC.

Similarly, Dr Selman discussed the molecular evidence explaining why some smokers develop COPD, others IPF, and others develop both. He suggested that this was due to a specific genetic architecture converging with specific epigenetic changes in a lung undergoing the normal effects of aging. This tied in with other important messages from the meeting: the role of aging and the evolution of the lung in the pathogenesis of many common respiratory disease (see our second message). Dr I. Buendía spoke about various genetic aspects of IPF in her presentation.

Dr Barbera explained the relationship between pulmonary hypertension (PH) and COPD. This phenomenon occurs in a significant number of COPD patients, particularly those with the most severe airflow limitations (GOLD 3–4), of whom 25%-50% have PH. In these patients, pulmonary vascular endothelial damage seems to play a fundamental role in the disease. Patients with both COPD and PH appear to have impaired circulating stem cell-derived repair mechanisms. These findings have led to an interest in the role of drugs acting on endothelial function in the treatment of combined COPD and PH.

Finally, Dr Barbé discussed the possible relationship between obstructive sleep apnea syndrome (OSAS) and cardiovascular diseases or cancer, pointing primarily to the lack of firm evidence that treating OSAS can reduce cardiovascular morbidity and mortality. Nevertheless, 2 clinical trials that may shed some light on the matter are currently underway. With regard to cancer, Dr Barbé cited emerging evidence, as yet unconfirmed, relating to both diseases. In any case, given the prevalence of the 3 diseases (OSAS, cardiovascular diseases, and cancer), this is an area of great interest for the healthcare community. In another oral presentation, Dr A. Sánchez discussed the effect of treating OSAS on the prognosis of acute coronary disease.
THE CONTRIBUTION OF ALTERED LUNG DEVELOPMENT AND AGING TO MANY ADULT RESPIRATORY DISEASES. CC16 MAY BE A USEFUL CLINICAL BIOMARKER

Dr. Fernando Martínez (Tucson, USA) discussed evidence indicating that the level of lung function achieved at the end of adolescence is a very important factor for indicating respiratory health in adulthood and old age, both for COPD and AB. This is due not only to genetic factors, but also to deficits acquired during the early years of life.

Drs. Agustí and Celli took a complementary perspective by discussing adults with COPD, and presented new evidence from their analysis of the Framingham, Copenhagen and Lovelance (New Mexico) cohorts. Their data clearly show that not all COPD patients follow the conventional pattern of accelerated loss of lung function with age in so-called “susceptible smokers”, described by Fletcher and Peto in the 1970s. Only about 50% of these patients show these characteristics, while in the others, changes in lung development in the early years of life (and this is the connection with the results of Dr. F. Martinez mentioned above), have a very significant effect on the appearance of COPD in adulthood.

It is interesting to note that both speakers identified CC16 as a good biomarker for lung development in both children and adults. CC16 (club cell 16, formerly Clara cell 16, and CC10 in mice, also known as secretoglobin 1A1, club cell secretory protein [CCSP], uteroglobin and urine protein-1) is a homodimeric protein, with a molecular weight of 15.8 kDa, secreted by club cells (nonciliated bronchiolar cells).

For his part, Dr. MacNee discussed the role of aging as one of the most significant risk factors for many diseases in humans, including respiratory diseases. He went so far as to suggest that many of them (including COPD) are in reality an acceleration of the normal process of lung aging. One of the most important factors in this setting is the balance between lung damage and repair. This balance is decisively affected by the accumulation of unrepaired molecular changes throughout life. A better understanding of these mechanisms may help prevent or delay the deleterious effects of aging on many aspects of human physiology.